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Issue Date: 03 August 2005

Case No. 1999-BLA-00834

In the Matter of

MARVIN DAN KINGERY,
Claimant

v.

CEMENTATION COMPANY OF AMERICA, INC.
Employer

GUNTHER- NASH MINING CONSTRUCTION COMPANY,
Employer

RANGER FUEL CORPORATION
Employer

DIRECTOR, OFFICE OF WORKERS COMPENSATION PROGRAMS,
Party In Interest

Appearances on Remand:

William D. Turner, Esq., for Claimant
Mary Lou Smith, Esq., for Gunther-Nash, Inc..
Dorothea J. Clark, Esq., for Ranger Fuel Corporation
Travis S. Haley, Esq., for Cementation Company of America, Inc.
Javier I. Romanach, Esq., for Director, OWCP

Before:

RICHARD E. HUDDLESTON
Administrative Law Judge

DECISION AND ORDER

This proceeding arises from a claim filed pursuant to the provisions of Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1972, and the Black Lung Benefits Reform Act of 1977, 30 U.S.C. § 901, et seq. (hereinafter referred to as the Act). Following a formal hearing, a decision and order awarding benefits (augmented by reason of one dependent, payable by Ranger Fuel Corporation, commencing October 1, 1995) was issued on November 26, 2002.

On appeal, the Benefits Review Board issued a decision and order on February 27, 2004, affirming in part, vacating in part, and remanding for further consideration. On July 1, 2004, the formal record was returned to this office by the Board. On September 14, 2004, an order was issued requiring briefs from all parties. On October 12, 2004, a brief was filed by Cementation Company of America, Inc. ("Cementation"). On October 13, 2004, briefs were filed by Gunther-Nash, Inc. ("Gunther")¹ and Ranger Fuel Corporation ("Ranger Fuel"). On November 1, 2004, a brief was filed by the Claimant. On November 1, 2004, Counsel for the Director, OWCP, submitted a copy of the brief it had filed with the Board and indicated his intent to rely upon the same.

ISSUES ON REMAND

The following issues have been remanded for resolution:

1. Which employer, if any, is the responsible operator for this claim;
2. Whether Claimant has pneumoconiosis as defined by the Act and regulations;
3. Whether Claimant is totally disabled;
4. Whether Claimant's total disability, if any, is due to pneumoconiosis.

DISCUSSION

In the Decision and Order issued November 26, 2002, Claimant was found to have fourteen and one-half years of coal mine employment. Gunther-Nash and Cementation were found not to meet the criteria of responsible operator, but Ranger Fuel was found to be the responsible operator liable for the payment of any benefits. Claimant was found to have established the existence of pneumoconiosis pursuant to 20 C.F.R. §718.202(a)(4), that he was entitled to the rebuttable presumption that his pneumoconiosis arose out of coal mine employment pursuant to 20 C.F.R. §718.203(b), and that the presumption was not rebutted. Claimant was found to be totally disabled under 20 C.F.R. §718.204(b)(2)(ii) and (iv), and that his total disability was due to pneumoconiosis pursuant to 20 C.F.R. §718.204(c). Accordingly, benefits were awarded.

The Board has affirmed, as unchallenged on appeal, the findings of fourteen and one half years of coal mine employment, no pneumoconiosis pursuant to 20 C.F.R. §718.202(a)(1)-(3), entitlement to the rebuttable presumption and no rebuttal of the presumption under 20 C.F.R. §718.203(b), and no total disability pursuant to 20 C.F.R. §718.204(b)(2)(i), (iii). Therefore, those findings will not be reconsidered.

¹ No explanation was given as to the corporate relationship between Gunther-Nash Mining Construction Company, and Gunther-Nash, Inc.

Responsible Operator

The Board has vacated the finding that Gunther could not be held as the responsible operator, and remanded the case for reconsideration of whether the evidence establishes that claimant was exposed to “coal mine dust,” and was, therefore, a miner, for more than one year while employed by Gunther pursuant to 20 C.F.R. §725.101(19), 725.202(b). *Kingery v. Ranger Fuel Corp.*, BRB No. 03-0270 BLA (February 27, 2004). Pursuant to the Board’s instructions on remand, both Gunther and Cementation must be considered as potential responsible operators in this claim. The Board instructed that it must first be considered whether the evidence establishes that Claimant was exposed to “coal mine dust” and thus was a miner for a cumulative period of one year while employed by Gunther. If it is determined that Gunther is not the responsible operator, the Board mandates consideration of whether Claimant was exposed to coal mine dust for a cumulative one year period while employed by Cementation.

A “responsible operator” is the operator that is liable for the payment of benefits to an eligible claimant for any period after December 31, 1973. 20 C.F.R. §725.493(a)(1) (2000). The regulations provide that the “responsible operator” is “the operator or other employer with which the miner had the most recent periods of cumulative employment of not less than one year.” 20 C.F.R. §725.493(a)(1) (2000).

As determined previously, the dates of Claimant’s employment with the three identified potential responsible operators in this case are as follows: Claimant worked in coal mine construction, as a welder and an electrician, for Ranger Fuel from September 27, 1965 to May 31, 1972. (Slip Op. at 4; DX 2, 3, 6; TR. at 36). Claimant then worked for Cementation in coal mine construction, as an electrician, from June 1, 1972 until May 1974. (Slip Op. at 4; DX 2, 3, 5; TR. at 36). Subsequently, Claimant worked for Gunther on two coal mine construction projects: the Maple Meadow site and the USX site. Claimant worked as a welder and an electrician for Gunther at the Maple Meadow site from October 27, 1983 to August 17, 1984, and as an electrician and a shot firer at the USX site from April 8, 1986 to December 23, 1986. (Slip Op. at 4; DX 2, 7; TR. at 37).

Claimant’s work for Gunther and Cementation involved coal mine construction, not coal mining. Under the Act, construction workers are only considered to be miners to the extent they were exposed to coal mine dust as a result of employment in or around a coal mine or coal preparation facility. However, such individuals are entitled to a rebuttal presumption that they were exposed to coal mine dust during all periods of such employment. The presumption may be rebutted by evidence demonstrating either of the following: (1) the individual was not regularly exposed to coal mine dust during his employment in or around a coal mine or coal preparation facility; or (2) the individual was not regularly employed in or around a coal mine or coal preparation facility. 20 C.F.R. § 725.202(a). This presumption is applicable both for determining whether the claimant was a miner in said employment, and for determining whether the employer is the responsible operator.

The Board remanded the previous finding that the presumption that Claimant was exposed to coal mine dust during his entire eighteen month tenure with Gunther was rebutted and that Gunther could not be held to be the responsible operator, since Claimant was exposed to

“coal dust” for a total of only nine of the eighteen months during which he worked for Gunther. The Board explained that, for purposes of determining whether Claimant worked as a “miner” for Gunther, the issue is not whether Claimant was exposed to “coal dust”, but whether Claimant was exposed to “coal mine dust” pursuant to 20 C.F.R. §725.202(b), which includes rock dust. Slip Op. at 6.

Claimant was employed by Gunther as an electrician on two construction projects. The first involved the construction of an air shaft for Maple Meadow Mining Company at a new mine site. (TR. at 37). The second project concerned Gunther’s construction of a slope at an existing mine site, Shawnee Mine on Pinnacle Creek, for the U.S. Steel Corporation, now known as USX. It has already been determined that Claimant was exposed to coal mine dust during his eight months of employment at the USX mining site, as well as for his last month at the Maple Meadow Mining site. 2002 ALJ Decision and Order at 7. Claimant specifically testified that he was not exposed to coal dust until the final month of his work at the Maple Meadow site, when the coal seam was reached, and that during his first nine months of work at the Maple Meadow site, he was exposed to “rock dust.” (TR. at 59-60). Claimant stated that he endured “rock dust exposure, from blasting, shooting rock,” down into the coal seam. (TR. at 45, 60). Because the Board specifically found that coal mine dust includes rock dust, and Claimant’s credible testimony of both rock dust and coal dust thus evidences a total of ten months of coal mine dust exposure at the Maple Meadows site.

I find that Claimant was exposed to rock dust while sinking the shaft at the Maple Meadow site. Pursuant to the Board’s instructions, this exposure constitutes coal dust exposure during coal mine employment at Gunther. The Board notes that the fact that the mine may not have been operational during this period is irrelevant, as a coal mine is defined as:

An area of land and all structure, facilities, machinery, tools, equipment, shafts, slopes, tunnels, excavations and other property, real or personal, placed upon, under or above the surface of such land by any person, used in, *or to be used in*, or resulting from, the work extracting in such area bituminous coal, lignite or anthracite from its natural deposits in the earth by any means or method, and in the work of preparing the coal so extracted, and includes custom coal preparation facilities.

20 C.F.R. § 725.101(a)(12) (emphasis added.) Constructing a shaft at a new mine is a task integral to the construction of a coal mine. Thus, these ten months of exposure in the Maple Meadow project, in addition to the eight months of coal dust exposure at the USX project, constitutes over a cumulative year of coal mine employment at Gunther. Gunther has offered no other evidence to rebut the presumption that Claimant was exposed to coal mine dust during his entire eighteen month tenure. As Gunther is the most recent employer, I find that Gunther is the operator responsible for this claim. 20 C.F.R. § 725.495.²

² The Board further found that “[i]f the administrative law judge finds on remand that Gunther-Nash is not the responsible operator, he must reconsider whether the evidence establishes that claimant was exposed to coal mine dust, and was, therefore, a miner, while working in mine construction for Cementation pursuant to Sections 725.101(19) and 725.202(b), and whether Cementation may properly be designated the responsible operator in this

Presence of Pneumoconiosis³

The Board affirmed the finding that pneumoconiosis was not demonstrated under § 718.202(a)(1). Additionally, as there is no autopsy evidence or biopsy evidence in the record and Claimant is not eligible for the enumerated presumptions, pneumoconiosis cannot be established under §§ 718.202(a)(2) or (a)(3). However, the Board remanded the matter for further consideration of whether Claimant has established the existence of pneumoconiosis under § 718.202(a)(4). Slip op. at 8.

Section 718.202(a)(4) provides for a finding of pneumoconiosis based upon the well-reasoned opinion of a physician who, while exercising sound medical judgment, finds, notwithstanding a negative x-ray, that the miner suffers from pneumoconiosis. This finding must be based on objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories.

The Benefits Review Board has held that the clause in this section “notwithstanding a negative x-ray” must be read to mean “even if there is a negative x-ray.” See *Taylor v. Director*, 9 BLR 1-22 BLA (1986). Thus, all physicians' reports must be considered, including those in which the physician's opinion is based in part upon a positive x-ray. In this case, there are medical reports of nine physicians in the record, three of which diagnosed pneumoconiosis.

Dr. Rasmussen

Dr. Donald L. Rasmussen is board certified in internal medicine. Dr. Rasmussen initially interviewed and examined Claimant on November 20, 1995 on behalf of the OWCP. Claimant provided a smoking history of one-half pack of cigarettes per day since 1962. Dr. Rasmussen's physical examination of Claimant revealed minimally decreased breath sounds, widespread low-pitched wheeze, and increased expiratory phase with forced respiration. Dr. Rasmussen cited an x-ray reading completed by Dr. Patel, an A-reader, which was positive for pneumoconiosis, 1/1. A pulmonary function study revealed a slight, irreversible obstructive ventilatory impairment. An arterial blood gas test showed a marked impairment in oxygen transfer during exercise. The SBDLCO⁴ was moderately decreased, the DL/VA minimally decreased. An electrocardiogram was within normal limits. (DX 14).

case.” This analysis is unnecessary as Gunther-Nash has been found to be the responsible operator in the present case.

³ A finding was made in the November 26, 2002 D&O that the overwhelming preponderance of x-ray evidence is negative for pneumoconiosis and the finding was thus made that pneumoconiosis was not demonstrated under § 718.202(a)(1). This finding has not been disturbed on remand.

⁴ This acronym is unexplained in Dr. Rasmussen's report, and research revealed no rational explanation. However, for the purposes of this opinion, it will be understood to represent the DCLO, single breath diffusing capacity for carbon monoxide results, referenced by Dr. Gaziano. See *infra*.

Following this evaluation, Dr. Rasmussen diagnosed coal workers' pneumoconiosis based on the occupational history and x-ray reading, due to coal mine dust exposure; and chronic bronchitis based on the chronic productive cough, due to both coal mine dust exposure and cigarette smoking. (DX 14). He concluded that Claimant "has marked loss of lung function. He is disabled from performing his last regular coal mine job." Dr. Rasmussen further stated:

[Claimant] has a long history of exposure to coal mine dust. He has x-ray changes consistent with pneumoconiosis. It is medically reasonable to conclude that he has coalworkers' pneumoconiosis which arose from his coal mine employment.

The two risk factors for [Claimant's] disabling respiratory insufficiency are his cigarette smoking and his coal mine dust exposure. The latter is the more significant factor since the pattern of impairment is that of an interstitial type lung disease.

(DX 14).

Dr. Rasmussen examined Claimant again on August 14, 1996, after Claimant came in complaining of shortness of breath on exertion and some rattling sensation in his anterior chest. (DX 67). Dr. Rasmussen noted that Claimant had a number of medical problems, including severe pulmonary insufficiency, and diffuse interstitial pulmonary fibrosis, secondary to occupational pneumoconiosis. Dr. Rasmussen's examination revealed a few rales over the left upper lobe anteriorly. He scheduled Claimant for pulmonary function studies, and additional tests. (DX 67).

Claimant returned on August 28, 1996 due to continued symptoms of shortness of breath with exertion. An electrocardiogram revealed sinus bradycardia, but was otherwise normal. Claimant's pulmonary function studies were normal without significant change after bronchodilator therapy. Claimant's maximum breathing capacity was normal. The single breath carbon monoxide diffusing capacity and the diffusing capacity normalized per liter alveolar volume (DL/VA) were moderately reduced. Resting arterial blood gases were abnormal with moderate impairment of oxygen transfer. (DX 67). Additionally:

[Claimant] underwent initially an incremental treadmill exercise study beginning at 2 mph at a 0% grade. This level was maintained for 3 minutes. Thereafter, the grade of the treadmill was increased 2.5% per minute. The patient exercised for 8 minutes and reached a maximum of 2 mph at a 12% grade. He achieved an oxygen uptake of 21 cc/kg/min. which is excessive for this exercise level. He was then allowed to rest for a period of time and was returned to the treadmill where he completed a constant work load exercise level again at 2 mph at a 12% grade for a 5 minute exercise stint. On this occasion, he achieved an oxygen consumption of 23.7 cc/kg/min. which is considerably in excess of this exercise level, and did amount to some 75% of his predicted maximum oxygen consumption. His heart rate was normal. He exceeded his anaerobic threshold normally at about 48% of his predicted maximum oxygen consumption. His

volume of ventilation was very markedly excessive and amounted to some 73% of his maximum breathing capacity indicating a maximum capacity for exercise. He also exhibited marked increase in VD/VT [dead space ventilation to tidal volume] ratio owing in part to an increase in respiratory frequency, but also associated with physiologic dead space of 1.18 liters per breath. This excessive dead space ventilation is a significant causative factor in his marked over-ventilation. Oxygen transfer was moderately impaired, with an A-a oxygen tension gradient rising to 35.3 mm Hg. His pO₂, however, was elevated because of his extreme over-ventilation which raised his alveolar oxygen tension to 113 mm Hg.

[Claimant] was in very marked distress following this exercise period. His base excess dropped to -9.9 and his bicarbonate dropped to 14, indicating a severely stressful exercise stint.

(DX 67).

Based upon these test results, Dr. Rasmussen concluded that:

[Claimant's] excessive ventilation is the consequence of his increased physiologic dead space which also correlates with his distinctly reduced single breath carbon monoxide diffusing capacity. [Claimant] has two primary causes for his disabling respiratory insufficiency. These are, of course, his cigarette smoking and his coal mine dust exposure. His coal mine dust exposure is by far the most significant factor in view of the fact that he has quite normal ventilatory function, but distinct disturbed gas exchange and reduced diffusing capacity.

(DX 67).

Dr. Rasmussen examined Claimant again on February 9, 2000, due to pain. The examination revealed a few fine inspiratory crackles, especially at the right base. Dr. Rasmussen found significant arterial insufficiency, prescribed medicine, asked for a cardiac consultation, and ordered further tests. (CX 1).

On February 17, 2000, Dr. Rasmussen interpreted Claimant's pulmonary function lab report:

Slight, irreversible obstructive ventilatory impairment. Maximum breathing capacity is normal. (Predicted 126). Single breath carbon monoxide diffusing capacity is markedly decreased. Minimal resting hypoxia.

(CX 1).

Dr. Gaziano

Dr. Dominic Gaziano is board certified in pulmonary medicine, and is a B-reader. At the request of the OWCP, Dr. Gaziano reviewed the medical records on July 18, 1997. He

concluded, based upon Claimant's pulmonary function tests (PFT) and Claimant's single breath pulmonary diffusing capacity for carbon monoxide (DLCO) results, that Claimant has pneumoconiosis. (DX 68). Dr. Gaziano specifically noted, "[C]laimant's p.f.t. 1995 and 1996 [completed by] Dr. Rasmussen results a moderately severe diffusion impairment that would be considered disabling for coal mine work. The recent assessments Dr. Rasmussen and Dr. Porterfield found evidence of occupational pneumoconiosis." (DX 68).

Dr. Porterfield

Dr. Porterfield is a pulmonary specialist who interviewed and examined Claimant on April 23, 1997 on behalf of the OWCP for a second opinion on Claimant's respiratory insufficiency. Dr. Porterfield was provided with the copies of the pulmonary function studies and cardiopulmonary stress test that had been obtained by Dr. Rasmussen. Dr. Porterfield noted that because he had copies of the results, he did not repeat these tests. (DX 64). Dr. Porterfield's physical examination of Claimant revealed that Claimant's lungs were "clear on all fields." Upon consideration of the available medical records, Dr. Porterfield concluded that:

Chest x-ray has demonstrated occupational pneumoconiosis P/p with a profusion of 1/1 in all lung zones. Spirometry performed by Dr. Rasmussen demonstrated normal flows with a very mild obstructive process demonstrated by slightly decreased FEV₁/FVC ratio. DLCO/VA was minimally decreased. The cardiopulmonary stress test demonstrated a maximum O₂ uptake of 23.6 cc's of O₂ per kilogram per minute. According to the AMA Guide to the Evaluation of Permanent Respiratory Impairment, this O₂ uptake with the cardiopulmonary stress test would place [Claimant] in a 10 to 25% mild impairment of the whole person.

Although it is impossible to say with certainty whether this is due to pneumoconiosis or cigarettes, the cigarette usage that this patient has engaged in, if correct, is less than 20 pack years and generally is not sufficient to produce a significant respiratory insufficiency. Thirty-four years in the mines has a much greater likelihood of doing this.

(DX 64).

Dr. Castle

Dr. Castle is an associate professor of medicine at the University of Virginia College of Medicine. (DX 46). He is board certified in pulmonary and internal medicine, and is a NIOSH certified B-reader. (DX 46, 92). Dr. Castle examined Claimant on two occasions.

Dr. Castle performed a pulmonary evaluation on Claimant on June 25, 1996. Claimant provided a smoking history of less than a pack of cigarettes per day since age thirty-two, giving him a less than thirty pack year smoking history. Dr. Castle also acknowledged that Claimant has credit for working for 14.53 years as a coal miner, and had worked as a federal mine inspector for sixteen years. (DX 46).

Dr. Castle's examination of Claimant's chest was normal. Dr. Castle reviewed an x-ray that was negative for coal workers' pneumoconiosis, 0/1. Dr. Castle further noted that Claimant's pulmonary function study was normal, as was the lung volumes. The diffusing capacity was mildly reduced consistent with the current smoking habit. Dr. Castle summarized, "[T]hese pulmonary function studies showed no evidence of obstruction and no restriction with a very mild reduction in the diffusing capacity related to his tobacco habit." (DX 46). Additionally, resting arterial blood gas test showed mild hypoxemia with a normal response to exercise. Specifically:

The pH was 7.440, the pCO₂ was 31.0 mmHg, the pO₂ was 65.7 mmHg. There was a normal response to exercise. The pO₂ increased. The pH at the end of the exercise was 7.420, the pCO₂ was 24.0 mmHg, the pO₂ was 70.2 mmHg.

(DX 46). A carboxyhemoglobin level⁵ was obtained and indicated the level of 5.6%, which Dr. Castle deemed compatible with smoking one pack of cigarettes per day. (DX 46). An electrocardiogram was also obtained, and showed no ischemic changes at rest or with exercise or thereafter. (DX 46).

After reviewing the aforementioned data, Dr. Castle rendered the following assessment:

1. No evidence of coal workers' pneumoconiosis by physical examination, radiographic evaluation, physiologic testing and arterial blood gases.
2. Tobacco smoke induced chronic bronchitis.
3. No respiratory impairment.
4. Mild hypoxemia which improves after exercise related to ventilation perfusion mismatching due to chronic bronchitis.
5. Elevated carboxyhemoglobin level of a current smoker.
6. History of hypertension.

(DX 46).

After reviewing additional medical records, including Dr. Rasmussen's 1995 report, Dr. Castle's diagnoses remained the same. Dr. Castle noted that Claimant had informed Dr. Rasmussen of a ½ pack a day smoking history, different from the less than 1 pack a day history

⁵ Dr. Castle explained in his deposition dated May 23, 2000, "A carboxyhemoglobin level is a measure of the hemoglobin in the blood that is tied up or bound with carbon monoxide. Carbon monoxide is a by-product of combustion, such as smoking cigarettes." (Ranger EX 14). Dr. Castle further noted:

[T]here is some correlation that indicates that first of all a nonsmoker should actually have a lower level, but in our society today, they may be around smoking or in an intimate relationship or whatever, so we consider anything less than 1.5 percent to be normal. About 4.5 to 5% may be seen in individuals, maybe 5.5 to 6%, who are smoking about a pack and a half of cigarettes daily. It will depend upon the frequency of their smoking and so forth, but 6 to 10 would be considered more than one or one and a half packs of cigarettes a day, maybe as much as two packs a day.

(Ranger EX 14).

he provided Dr. Castle. Dr. Castle opined, “Nevertheless, a 16-pack-year smoking history is significant enough exposure in a susceptible host to have caused him to develop chronic obstructive pulmonary disease, i.e. chronic bronchitis and/or emphysema or lung cancer in a susceptible host.” (DX 46).

Dr. Castle explained that Claimant’s physical exam did not demonstrate symptoms associated with interstitial pulmonary process, as it lacked the finding of rales, crackles or crepitations. Dr. Castle acknowledged that wheezing and reduced airflow was noted in Dr. Rasmussen’s examination, and opined that these findings “are associated with tobacco induced bronchitis.” (DX 46). Dr. Castle found no evidence of obstruction in the physiologic studies, and Claimant’s lung volumes were normal in his examination, indicating no evidence of restriction. Claimant had a very mild reduction in his diffusing capacity, which Dr. Castle explained as “a commonplace finding in patients that are tobacco users.” (DX 46).

Dr. Castle acknowledged that Claimant’s arterial blood gases showed a mild degree of hypoxemia, but that:

[I]t is my opinion that this hypoxemia is related to ventilation perfusion, mismatching brought on by his tobacco smoke induced bronchitis. If coal workers’ pneumoconiosis causes hypoxemia, it is irreversible and does not improve with time or treatment. It also causes a fall in the oxygen tension with exercise. That was not the finding in this case. In fact, [Claimant’s] pO_2 improved with exercise, which is a normal response to exercise.

(DX 46). Dr. Castle thus opined to a reasonable degree of medical certainty that Claimant does not suffer from coal worker’s pneumoconiosis and has no significant respiratory impairment whatsoever.

Dr. Castle examined and interviewed Claimant again on August 27, 1997. Claimant’s smoking history was one-half pack of cigarettes per day for thirty-four years. Examination was essentially unremarkable, thereby correcting the previous history obtained. An x-ray dated 8/27/92 was read as 0/1, t/s, mid and lower zones. Dr. Castle noted evidence of cardiomegaly, but noted that there were no changes consistent with coal workers’ pneumoconiosis. Dr. Castle observed that Claimant’s pulmonary function studies showed a mild, clinically insignificant airways obstruction and a reduction in the diffusing capacity. Claimant’s lung volumes were normal indicating no restriction. Claimant’s resting arterial blood gas test was normal, but after exercise, the test displayed a mild degree of hypoxemia. The carboxyhemoglobin level was elevated at 4.5%.⁶ (DX 92).

⁶ Dr. Castle was deposed on May 23, 2000. He testified that a carboxyhemoglobin level of 4.5% could be consistent with someone smoking around a half a pack of cigarettes per day, but that it is likely that that level represents “someone smoking more than that, depending upon how long it had been since they smoked their last cigarette.” He further stated that “[t]he elevation of the carbon monoxide level to 4.5 percent would certainly affect [the diffusion capacity] to some degree.” (Ranger EX 14).

After reviewing all of the data obtained at the time of examination, Dr. Castle rendered the following assessment:

1. No evidence of coal workers' pneumoconiosis by physical examination, radiographic evaluation and physiologic testing.
2. Chronic bronchitis, tobacco smoke induced.
3. Mild, clinically insignificant airways obstruction with reduction in diffusing capacity, tobacco smoke induced.
4. Hypertension with cardiomegaly.
5. History of skin cancer.
6. Elevated carboxyhemoglobin level consistent with current smoking habit.

(DX 92).

Dr. Castle then reviewed other medical records, including his previous report. As to Dr. Rasmussen's report, Dr. Castle remarked that:

During exercise an oxygen uptake of 21.9 cc/kg/minute was obtained. Dr. Rasmussen indicates that this is excessive for this level of exercise. He states that it is probably related to increased work of breathing secondary to his overventilation. Dr. Rasmussen does not note that during test 1 [Claimant] had a marked hypertensive response. He also does not note that [Claimant] developed a very significant metabolic acidosis during the exercise study. These findings are indicative of cardiovascular limitations to exercise.

(DX 92).

Upon review of the medical data, Dr. Castle concluded that Claimant does not have coal workers' pneumoconiosis based on a variety of factors, including the lack of examination findings of rales, crackles, or crepitations on a consistent basis; the negative x-ray readings; the very minimal, clinically insignificant airways obstruction; the normal lung volumes; the minimal reduction in diffusing capacity which occurred from 1995 to 1997 while Claimant continued to smoke, but not mine coal; and the mild degree of hypoxemia with exercise being related to a cardiovascular limitation to exercise. Dr. Castle pointed out that the findings on the exercise study, "in conjunction with his history of chest pain with radiation to both his left arm and jaw, suggest the possibility of angina pectoris associated with hypertensive cardiovascular disease." Dr. Castle found no respiratory or pulmonary disability. (DX 92).

Dr. Castle issued a supplemental report on April 26, 2000, stating that his opinions remained the same. (Ranger EX 12). Dr. Castle additionally noted in this report that Claimant now had documented evidence of hypertension and atherosclerotic vascular disease. Dr. Castle explained:

[Claimant] had a carotid ultrasound which showed evidence of peripheral vascular disease and he had a Cardiolute stress test which showed evidence of a previous myocardial infarction with hypokinesis and reduced ejection fraction, indicating

reduced function of the left ventricle. These findings clearly can cause both symptomatic shortness of breath as well as arterial blood gas abnormalities.”

(Ranger EX 12). Dr. Castle then concluded, “It was my opinion in the past, and remains my opinion that this decline [in pO₂] and this variable change in the pO₂ with exercise is related to his underlying coronary artery disease and left ventricular dysfunction.” (Ranger EX 12).

Dr. Zaldivar

Dr. George L. Zaldivar is a clinical professor of medicine at the West Virginia University School of Medicine. (Ranger EX 8). Dr. Zaldivar is board certified in internal, pulmonary, critical care, and sleep disorder medicine, and is a NIOSH certified B-reader. (Ranger EX 8).

Dr. Zaldivar interviewed and examined Claimant on September 25, 1996 on behalf of an employer, and reviewed Dr. Castle’s results. Dr. Zaldivar also noted minimal airway obstruction by spirometry, normal resting exercise blood gases, normal lung volumes, moderate diffusion impairment from undetermined cause, and radiographic evidence of pneumoconiosis.

(CX 1). Dr. Zaldivar elaborated in his report:

After reviewing the results obtained by Dr. Castle, I find that the ventilatory study of [Claimant] hovers between minimal airway obstructions as it was present in my office and normal spirometry as was found by Dr. Castle. [Claimant] still smokes and therefore this minute variability in his spirometry is understandable and it is caused by inflammation of the airways due to cigarette smoking.

It is my opinion that [Claimant] does have radiographic pneumoconiosis. This pneumoconiosis however has not resulted in any pulmonary impairment. The moderate diffusion abnormalities found in my office is in all likelihood due to smoking. Smoker’s (sic) invalidate their diffusing capacity study because physiologically there are several mechanisms by which they artificially reduce their diffusion. Cessation of the smoking causes the diffusing capacity to improve over time.

The diffusion abnormality which I found in my office did not cause any problem with blood oxygenation. His resting and exercise blood gasses (sic) were both normal.

He found no disability. (CX 2).

On May 12, 1997, Dr. Zaldivar wrote to the employer that:

I have reviewed the reports of the chest x-rays which you sent me. I notice that most readers have found some evidence of abnormality in the chest x-ray, some of these readers have attributed it to a form of pneumoconiosis, and some to other causes. The films are not normal. It was my interpretation of my films that the

findings were compatible with a form of pneumoconiosis which in this case would be due to his occupation. I believe that all of us are looking at the same densities and given it different interpretation to what all of us are seeing.

(CX 2).

Dr. Zaldivar issued a supplemental report on April 23, 2000 upon receiving additional medical records regarding Claimant. Dr. Zaldivar opined that the records displayed that Claimant had a low diffusion capacity for several years. Dr. Zaldivar explained that Claimant's blood gases had been well preserved with exercise, until Dr. Castle's examination, in which they dropped for the first time during exercise. (Ranger EX 11). Dr. Zaldivar recollected, "In 1996 when I examined him the blood gases were normal at rest and with exercise in spite of the low diffusion capacity, which I attributed to smoking." (Ranger EX 11).

Dr. Zaldivar further recalled previously interpreting Claimant's chest x-ray as showing pneumoconiosis. In his 2000 report, Dr. Zaldivar acknowledged that "the pneumoconiosis was very early and other readers have read the x-rays as it showing nonspecific interstitial fibrosis which may well be accurate, particularly with a low diffusion capacity." (RX 11).

Dr. Zaldivar concluded:

Given the smoking history in this case and the minimal changes in the chest x-ray, my opinion is that emphysema from smoking is responsible for reduced diffusion. The deterioration of the blood gases when tested by Dr. Castle is due to the fact that now [Claimant] has developed cardiac disease with left ventricular failure as demonstrated by Dr. Barghouthi in his exercise test. The combination of poor cardiac function and ventilation and perfusion mismatch caused by the emphysema has resulted in the problem with the blood gases during the last test. None of these changes are due to pneumoconiosis. Cardiac disease is not caused by coal workers pneumoconiosis, nor is emphysema.

(Ranger EX 11).

Dr. Spagnolo

Dr. Samuel V. Spagnolo is a professor of medicine at George Washington University School of Medicine and Health Services. (Ranger EX 8). He is board certified in internal and pulmonary medicine, and has authored numerous publications on lung and pulmonary diseases. (Ranger EX 8). Dr. Spagnolo performed a record review on behalf of an employer on June 10, 1999. Upon conclusion of his review, Dr. Spagnolo concluded that Claimant had neither coal workers' pneumoconiosis, nor a pulmonary/respiratory impairment attributable to pneumoconiosis. He concluded that Claimant does not have coal workers' pneumoconiosis based on the negative physical examinations for evidence of lung disease; the multiple negative chest x-rays, and the multiple normal or nearly normal tests of lung function. (Ranger EX 3).

Dr. Spagnolo noted that Claimant had a slightly reduced FEV₁/FVC noted in November 1995, but that it was normal in 1997 when tested by Dr. Castle. Dr. Spagnolo opined that this pattern indicates “intermittent, mild, and reversible airflow obstruction. [Claimant’s] long exposure to continuous cigarette smoking is the reason for this phenomena. Chronic, continuous cigarette abuse is also the reason for his chronic productive cough.” (Ranger EX 3). Dr. Spagnolo also noted that the most recent tests of Claimant’s lung function, as of the date of his report, demonstrate a normal total lung capacity (TLC), normal forced vital capacity (FVC), normal FEV₁, and normal FEV₁/FVC. Dr. Spagnolo concluded that there is no evidence of either an obstructive or restrictive lung impairment. “Thus, there is no basis for a diagnosis of emphysema or an interstitial lung disease such as a pneumoconiosis.” (Ranger EX 3).

Dr. Spagnolo also stated that:

In the setting of normal spirometry, normal lung volumes and a negative chest x-ray, the isolated reduction in the PaO₂ and DLCO is best explained by pulmonary vascular changes induced by the calcium channel blocker Norvasc. I have seen this drug effect on numerous occasions and it can be easily confirmed by withdrawing the calcium channel blocker and repeat testing. This could be very important to [Claimant’s] long term health since other agents can easily be substituted for a calcium channel blocker for control of his blood pressure.

(Ranger EX 3).

Upon his review of additional medical records, Dr. Spagnolo issued a supplemental opinion dated April 22, 2000. Dr. Spagnolo indicated that his opinions remained the same, specifically stating:

[Claimant] has no evidence of an interstitial lung condition such as pneumoconiosis by physical examination, pulmonary function testing or chest radiograph. He does not have a pulmonary/respiratory impairment attributable to pneumoconiosis.

(RX 13). Dr. Spagnolo noted that Claimant’s FEV₁/FVC was reduced in 1995 and 2000, but was normal in 1997. Dr. Spagnolo explained, “This pattern can be observed in healthy individuals and an obstructive dysfunction should not be inferred solely by the reduced FEV₁/FVC.” (RX 13). Dr. Spagnolo also observed that “[t]he arterial blood gas values are also normal in the supplemental material which is now available. This finding may be related to the withdrawal of the calcium channel blocker, Norvasc.” (Ranger EX 1). Dr. Spagnolo opined in his supplemental report:

In the setting of normal spirometry, normal lung volume, normal blood gas values and a negative chest x-ray, the isolated reduction in the DLCO test is best explained by non-specific airway changes related to continued cigarette smoking. I would agree with Dr. W.K.C. Morgan’s comments with regard to this medical phenomena and I agree with him that the DLCO should not be used as a screening test for pneumoconiosis in anyone who is a chronic cigarette smoker.

(Ranger EX 13). Dr. Spagnolo concluded, Claimant's "chronic long-term cigarette smoking and chronic heart disease are responsible for his medical complaints. None of these symptoms are related to or associated with coal dust exposure." (Ranger EX 13).

Dr. W. K. C. Morgan

Dr. Morgan holds the British equivalent of the American board certification in internal medicine, and is a NIOSH certified B-reader, with years of experience examining coal miners. (Ranger EX 4). Dr. Morgan has written numerous publications regarding pneumoconiosis. (*Id.*). Upon request of an employer, Dr. Morgan reviewed Claimant's medical records, and issued a report dated June 21, 1999.

Upon conclusion of his review, Dr. Morgan opined that Claimant has "an absolutely normal ventilatory capacity." (Ranger EX 4). Dr. Morgan explained that each time Claimant's ventilatory capacity was measured it was better than normal, and there was no change in the bronchodilators. Dr. Morgan noted that Claimant's initial arterial blood gas study showed a low PO₂ but noted that when Claimant seen by Dr. Castle, his PO₂ was normal. Dr. Morgan stated that Claimant's diffusing capacity is "probably slightly to moderately reduced." Dr. Morgan opined that "[b]oth the abnormalities of [Claimant's] blood gases and diffusing capacity are best accounted for by the fact that [Claimant] has emphysema." (Ranger EX 4).

Dr. Morgan further opined that Claimant did not suffer from coal workers' pneumoconiosis, and did not believe that Claimant's exposure was sufficient to produce this condition. Dr. Morgan explained:

Even though [Claimant] may have spent 15 years underground, most of this time was not spent in dusty areas nor was it spent at the coal face. [Claimant's] subsequent time in the mines as a Federal Health and Safety Inspector would not expose him to large quantities of coal dust since for the most part he would be well away from the coal face.

(Ranger EX 4). Dr. Morgan also cited in support the several negative readings completed by noted B-readers. (Ranger EX 4).

Dr. Morgan was also of the opinion that Claimant was not forthcoming about his smoking history. Dr. Morgan explained:

Moreover, when Mr. Kingery's carboxyhaemoglobin level was tested by Dr. Castle and also by Dr. Rasmussen it was over 5% and on a third occasion it was down to 4.5%. If one takes into consideration that Mr. Kingery had been sitting for one or two hours and undergoing lung function tests and not being able to smoke, a carboxyhaemoglobin level of 5% means that he was smoking at least 1 1/2 packages of cigarettes a day. In short, I do not find Mr. Kingery's cigarette smoking histories accurate.

(Ranger EX 4).

Dr. Fino

Dr. Fino is board certified in internal and pulmonary medicine, and is a NIOSH B-reader. (DX 53, 111). Upon reviewing Claimant's medical records, Dr. Fino issued a report on February 10, 1999. Dr. Fino opined that Claimant has normal spirometry, as his FVC, FEV₁ and MVV values are normal. Dr. Fino explained:

The 11/20/95 lung function study showed a slight reduction in the ratio of the FEV₁ to the FVC suggesting an obstruction abnormality. However, there was no impairment considering the normal FEV₁. The 6/25/96 and 8/27/97 pulmonary function studies did not show any evidence of obstruction.

During all of the above lung function studies, [Claimant] was smoking. He had worked for a total of 15 1/2 years in the mines and subsequently worked as a federal coal mine inspector, retiring from that job in 1996.

The variable obstruction (that is, the obstruction seen on one lung function study but not on the others) is consistent with smoking and not consistent with a coal mine dust related condition.

[Claimant] has had reduced diffusing capacities on all of the studies that were performed. I noted elevated carboxyhemoglobin levels in the records, and the patient was actively smoking during these studies. Active cigarette smoking is a common cause of a reduction in diffusion. Therefore, I would attribute his reduction in diffusion to smoking.

[Claimant] had three exercise tests. The first two exercise tests showed no decrease in the blood oxygen level with exercise. There was some hyperventilation on these two studies, and the carbon dioxide levels were on the low side. There was also an elevation in the alveolar-arterial oxygen gradient. This was an abnormal exercise study since the pO₂ value normally would not drop with exercise as it did in this particular case.

Dr. Castle brought up the possibility of coronary artery disease based on this man's history of chest pain with radiation into the arm.

I believe that there is a variable abnormality in oxygen transfer in this case which is consistent with his continued smoking. I cannot attribute this to a coal mine dust related pulmonary condition because it has not been uniformly present throughout the several exercise tests.

(DX 111).

Dr. Fino issued a supplemental report on April 18, 2000 after reviewing additional medical evidence. His conclusions remained the same. (Ranger EX 7).

Dr. Jarboe

Dr. Jarboe is board certified in internal and pulmonary medicine, and is a NIOSH certified B-reader. Dr. Jarboe reviewed Claimant's medical records upon the request of Ranger Fuel after which he issued a report of his findings dated February 9, 1999. (DX 111).

Upon review of Claimant's medical records, Dr. Jarboe opined that there was not sufficient objective evidence to diagnose coal workers' pneumoconiosis. Dr. Jarboe cited the lack of radiographic evidence, and noted a number of negative readings by highly regarded B-readers. Dr. Jarboe further opined that there was a lack of physiologic evidence to make a diagnosis. Dr. Jarboe explained:

When coal dust inhalation causes impairment, it usually does so by reducing FVC and FEV₁. In the most recently available spirogram of 8/27/97, both FVC and FEV₁ were completely normal. There was no restriction or obstruction. If [Claimant] had a dust induced lung disease, I would anticipate some reduction in both FVC and FEV₁.

(DX 111).

Dr. Jarboe was also asked to offer an opinion as to whether Claimant has any pulmonary or respiratory impairment, to which he responded:

[Claimant] does have a mild to moderate respiratory impairment. He has no impairment based on spirometric testing. However, on his most recent diffusion capacity there was a mild to moderate reduction. This could be accounted for at least in part by his cigarette smoking and elevated carbon monoxide levels.

[. . .] [T]his mild to moderate reduction in diffusion is the result of pulmonary emphysema and not coal workers' pneumoconiosis. I base this statement on the following reasoning. First, there is no evidence in the chest radiograph that [Claimant] has any significant dust retention in his lungs. Second, Dr. Scott read the x-ray of 6/25/96 as showing emphysema. Dr. Castle measured the residual volume on 6/25/96 as showing emphysema. Dr. Castle measured the residual volume on 6/25/96 as 125% of normal. This combination of radiographic findings of emphysema coupled with a reduced diffusion capacity and an elevated residual volume constitutes adequate evidence of this disorder.

It is my opinion that this pulmonary emphysema has been caused by cigarette smoking and not dust inhalation. There is evidence of a significant smoking history. While Mr. Kingery claims to have consistently used less than a package of cigarettes a day, the objective evidence would indicate that he smokes over a pack a day. This has been shown by the carboxyhemoglobin levels measured by

Dr. Castle on two separate occasions. Based on the evidence, it would appear that the claimant has smoked over a pack a day for over 30 years, a significant smoking history that could readily cause mild to moderate emphysema.

Coal dust inhalation does cause mild focal emphysema but in the absence of significant dust retention, more severe forms of emphysema do not develop. Since [Claimant] has no evidence of significant dust retention of the chest radiograph, it is my reasoned opinion that his emphysema can be ascribed to smoking.

(DX 111).

Analysis

In weighing conflicting opinions, an undocumented or unreasoned opinion may be given little or no weight. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(en banc). A “documented” opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). A “reasoned” opinion is one in which the judge finds the underlying documentation and data adequate to support the physician’s conclusions. *Fields, supra*. An opinion may be adequately documented if it is based on items such as physical examinations, symptoms, and the patient’s work and social histories. *Hoffman v. B&G Construction Co.*, 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984); *Justus v. Director, OWCP*, 6 B.L.R. 1-1127 (1987). Whether a medical report is sufficiently documented and reasoned is for the judge as the finder of fact to decide. *Clark, supra*. As instructed by the Board, I must address the relevant factors bearing on the credibility of the medical opinions, including the qualifications of the respective physicians, the explanation of their medical opinions, the documentation underlying their medical judgment, and the sophistication and basis of their diagnoses. *See Milburn Colliery Co. v. Hicks*, 138 F.3d 524, 21 BLR 2-323 (4th Cir. 1998); *Sterling Smokeless Coal Co. v. Akers*, 131 F.3d 438, 21 BLR 2-269 (4th Cir. 1997); Slip Op. at 9. Additionally, if it is found on remand that the medical opinion evidence is sufficient to establish the existence of pneumoconiosis under Section 718.202(a)(4), all relevant evidence must be weighed together, like and unlike, under Section 718.202(a)(1) – (4) prior to making the ultimate determination as to whether Claimant has established the existence of pneumoconiosis under Section 718.202(a). *Compton*, 211 F.3d at 208-209, 22 BLR at 2-170, Slip Op. at 5.

At the outset, I find that Dr. Morgan’s opinion is entitled to no weight, as I find it to be more speculative than well-reasoned or well-documented. Highly suspect is Dr. Morgan’s opinion that while Claimant “may have spent 15 years underground,” such exposure was insufficient to produce coal worker’s pneumoconiosis in a susceptible host, and that “most of this time was not spent in dusty areas nor was it spent at the coal face.” (Ranger EX 4-10). Additionally confusing is Dr. Morgan’s statement that Claimant’s job as a mine inspector was “not [. . .] a particularly arduous job.” (Ranger EX 4-10). Claimant credibly testified about the conditions of his working environment. Specifically, Claimant testified that throughout practically all of his work history in the mines, which was affirmed as 14.5 years in length, he was exposed to coal dust or dusty conditions. (TR. at 38). In contrast, Dr. Morgan provided no

evidence or basis of justification for his statement that most of Claimant's employment was not spent in dusty areas. As a portion of his medical opinion is based upon such blatant speculation, I find that Dr. Morgan's opinion is not entitled to weight.

Dr. Spagnolo's medical opinion is also unreliable. Dr. Spagnolo concluded that Claimant's isolated reduction in pO_2 and single breath pulmonary diffusing capacity for carbon monoxide is best explained by pulmonary vascular changes induced by the calcium channel blocker Norvasc. (Ranger EX 3). However, Dr. Spagnolo questioned the reduction in the pO_2 to begin with, suspecting that the resting and exercise pO_2 values were reversed, a suspicion not borne out. On a subsequent test completed by Dr. Rasmussen on February 17, 2000, when Claimant supposedly was not on Norvasc, the arterial blood gas test nevertheless produced qualifying values.⁷ (CX 1). Not only did Dr. Spagnolo deem the results from this 2000 test "normal," he also associated these "normal" results to the withdrawal of the calcium channel blocker, Norvasc. (Ranger EX 1). However, as affirmed by the Board, the results were not normal. Therefore, because the reasoning underlying Dr. Spagnolo's opinion is questionable, I give Dr. Spagnolo's opinion little weight.

Dr. Porterfield's opinion that Claimant has a mild impairment due to pneumoconiosis is entitled to no weight because his diagnosis is based solely upon Dr. Rasmussen's notation of a positive x-ray reading. (DX 64). This diagnosis is contrary to the overwhelming weight of the x-ray evidence that is negative for pneumoconiosis, a finding that has been affirmed by the Board. While Dr. Porterfield also acknowledges in his report Claimant's pulmonary function studies and blood gas studies, his report fails to explain why the results from these tests would support a diagnosis of either clinical or legal pneumoconiosis. Additionally, Dr. Porterfield's report is silent as to whether he would diagnose pneumoconiosis after considering the overwhelming negative x-ray evidence. Because Dr. Porterfield's opinion is unexplained and against the weight of the objective medical evidence, I find that his report is entitled to no weight.

Additionally unreliable is the opinion of Dr. Gaziano, who seconded the pneumoconiosis diagnoses of Drs. Rasmussen and Porterfield upon conducting a review of Claimant's medical records. There is no indication that Dr. Gaziano considered either Claimant's smoking history or his coal dust exposure history. It is also unclear from the record if Dr. Gaziano considered the additional objective data, such as Claimant's blood gas studies, in rendering his opinion. In concluding that Claimant suffers from coal workers' pneumoconiosis, Dr. Gaziano merely relied upon the diagnosis of pneumoconiosis of Drs. Porterfield and Rasmussen, which is entitled to no weight because it is based solely upon x-ray evidence that is against the preponderance of negative readings. Dr. Gaziano additionally noted that Claimant has "moderately severe diffusion impairment," though he never positively linked this condition to either Claimant's coal dust exposure or his smoking history. (DX 68). Because Dr. Gaziano failed to sufficiently

⁷ The February 17, 2000 arterial blood gas testing, completed by Dr. Rasmussen, yielded a pCO_2 level of 30.0 and a pO_2 level of 17. Though I am unable to ascertain precisely the altitude at which this test was performed, the Board affirmed that the results from this 2000 blood gas study as producing qualifying results for total disability. Slip Op. at 9.

explain his opinion, and appears to have considered only limited objective data, I find that his diagnosis of pneumoconiosis is entitled to less weight.

The remaining medical opinions seemingly agree that Claimant suffers from a lung impairment, though they diametrically disagree whether this condition arises from Claimant's coal dust exposure or solely from his tobacco usage.⁸

In addition to clinical coal workers' pneumoconiosis, Dr. Rasmussen diagnosed chronic bronchitis, disturbed gas exchange, and reduced diffusing capacity, all due to both cigarette smoking and coal dust exposure. At the outset, Dr. Rasmussen's finding of coal worker's pneumoconiosis is discredited because it was based primarily on x-ray evidence. As previously stated, and as affirmed by the Board, the overwhelming preponderance of the x-ray evidence is negative for clinical pneumoconiosis. (DX 14). However, Dr. Rasmussen found that Claimant suffers from chronic bronchitis, which he positively linked to Claimant's coal mine dust exposure, because it is more significant than his smoking "in view of the fact that he has quite normal ventilatory function, but distinct disturbed gas exchange and reduced diffusing capacity." (DX 67).

⁸ To reiterate, I have previously found that Claimant has failed to establish by a preponderance of the evidence the existence of clinical pneumoconiosis. However, the issue remains whether he has established the presence of legal pneumoconiosis. The Act itself defines pneumoconiosis as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 30 U.S.C. § 902(b). The regulations recognize that the Act's definition "includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis." 20 C.F.R. § 718.201(a) (internal quotation marks in original). Further, the regulations provide the following comprehensive definition of "clinical" and "legal" pneumoconiosis:

(1) Clinical Pneumoconiosis: "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.

(2) Legal Pneumoconiosis: "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. Definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

(b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

(c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure. 20 C.F.R. § 718.201.

Physicians do not always make a clear distinction in their reports and testimony between "clinical" and "legal" pneumoconiosis, and the Court of Appeals for the Fourth Circuit had repeatedly admonished ALJs and the Board to "bear in mind when considering medical evidence that physicians generally use 'pneumoconiosis' as a medical term that comprises merely a small subset of the afflictions compensable under the Act." *Barber v. Director, OWCP*, 43 F.3d 899, 901 (1995) (quotation marks and italics in original).

Dr. Jarboe related Claimant's mild to moderate reduction in diffusion capacity to emphysema from smoking, with at least part of the reduction possibly also due to the continuing cigarette smoking and consequent elevated carbon monoxide levels.

Dr. Castle found mild hypoxemia related to ventilation perfusion mismatching due to chronic bronchitis, and a reduction in the diffusing capacity, all due to cigarette smoking.

Dr. Fino attributed all of Claimant's reduction in diffusing capacity to active cigarette smoking. He stated that the variable abnormality in oxygen transfer is also consistent with continued smoking.

While Dr. Zaldivar initially diagnosed clinical coal workers' pneumoconiosis based on the x-ray reading, he later changed this opinion and related Claimant's mild airway obstruction to cigarette smoking and the moderate diffusion impairment to an undetermined cause, likely smoking.

In weighing these opinions, I find that Claimant has established that he suffers from emphysema and chronic bronchitis, both subcategories of chronic obstructive pulmonary disease (COPD). However, I find that the preponderance of the evidence in the record fails to establish that these conditions arise out of Claimant's coal mine employment.

Claimant's smoking history has been questioned in determining the catalyst of his COPD. Both Dr. Jarboe and Dr. Castle suggest that Claimant's smoking history is more significant than that to which he testified, and point to objective medical evidence in support of this conclusion. Dr. Castle performed a blood gas study upon Claimant on August 27, 1997, and obtained a carboxyhemoglobin level of 4.5%. Dr. Castle testified that a carboxyhemoglobin level of 4.5% likely represents someone smoking more than one-half pack a day. Dr. Jarboe supported this opinion by stating, "it would appear that [Claimant] has smoked over a pack a day for over 30 years, a significant smoking history that could readily cause mild to moderate emphysema." (DX 111). Additionally, Dr. Castle highlighted that the most recent results were from an examination of Claimant performed by Dr. Rasmussen on February 17, 2000. These results indicated that Claimant's carboxhemoglobin level was 7.0%. Dr. Castle again noted that this "is a very high level carboxyhemoglobin indicating someone smoking far in excess of ½ pack of cigarettes daily." (Ranger EX 12). Dr. Fino also highlights an elevated carboxyhemoglobin level. (DX 111). Notably, Dr. Rasmussen neglects to discuss his opinion of Claimant's carboxyhemoglobin level, and Claimant has failed to offer additional evidence into the record that could possibly explain his elevated carboxyhemoglobin level.

Further, though Claimant testified credibly at the hearing that he smoked a half a pack a day and has been relatively consistent in his other medical records, there is evidence in the record that he provided Dr. Castle with a more significant smoking history. (DX 46). This inconsistency questions the reliability of Claimant's testimony, especially in light of objective medical evidence tending to suggest that Claimant actually has a greater smoking history than that to which he testified. Regardless of the objective medical evidence and Claimant's inconsistency, Dr. Castle opined unequivocally, "Nevertheless, a 16-pack-year smoking history [associated with a ½ a pack a day smoking history] is significant enough exposure in a

susceptible host to have caused him to develop chronic obstructive pulmonary disease, i.e. chronic bronchitis and/or emphysema or lung cancer in a susceptible host.” (DX 46). Therefore, relying on a more significant smoking history does not affect the credibility of the medical opinions of Drs. Castle and Jarboe.

To be entitled to benefits under the Act, Claimant must prove by a preponderance of the evidence that his lung impairments are chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in his coal mine employment. The only physician of record to positively diagnose legal pneumoconiosis is Dr. Rasmussen. I find that this medical opinion fails to establish by a preponderance of the evidence the presence of legal pneumoconiosis, especially when considering the contradictory opinions of Drs. Jarboe, Castle, Fino and Zaldivar. In finding that Claimant’s respiratory ailments were substantially related to his coal dust exposure, Dr. Rasmussen stated “[Claimant] has two primary causes for his disabling respiratory insufficiency. These are, of course, his cigarette smoking and his coal mine dust exposure. His coal mine dust exposure is by far the most significant factor in view of the fact that he has quite normal ventilatory function, but distinct disturbed gas exchange and reduced diffusing capacity.” (DX 67). However, beyond this conclusory statement, there is no further supportive explanation in the record. It is thus unclear why, in Dr. Rasmussen’s opinion, the distinct disturbed gas exchange and reduced diffusing capacity would force the conclusion that the significant catalyst of Claimant’s COPD is his coal dust exposure, thus entitling him to benefits under the Act.

The other credible medical opinions in the record offer a vastly different explanation of Claimant’s distinct disturbed gas exchange⁹ and reduced diffusing capacity. In 1999, Dr. Fino stated that he believed there is a “variable abnormality in oxygen transfer in this case which is consistent with his [Claimant’s] continued smoking.” Dr. Fino explained that this is unrelated to Claimant’s coal mine dust exposure “because [this exposure] has not been uniformly present throughout the several exercise tests.” (DX 111). Drs. Castle and Zaldivar opined that Claimant’s arterial blood gas abnormalities seen most recently in his 2000 test are caused by his cardiac problems.¹⁰ Dr. Zaldivar elaborated, “The combination of poor cardiac function and ventilation and perfusion mismatch caused by the emphysema has resulted in the problem with the blood gases during the last test. None of these changes are due to pneumoconiosis. Cardiac disease is not caused by coal workers pneumoconiosis, nor is emphysema.” (Ranger EX 11).

⁹ Claimant’s arterial blood gas tests produced mixed results, as determined by applying the Table at Appendix C to Part 718. The November 20, 1995 study was qualifying at rest (pCO₂ level of 34 and pO₂ level of 64), non-qualifying with exercise (pCO₂ level of 26, and pO₂ level of 75). (DX 15). The June 25, 1996 study was qualifying both at rest (pCO₂ level of 31.0 and pO₂ level of 65.7) and with exercise (pCO₂ level of 24 and pO₂ level of 70.2). (DX 46). The August 28, 1996 study was qualifying at rest (pCO₂ was 35 and pO₂ was 65), non-qualifying with exercise (pCO₂ 30 and pO₂ 74; pCO₂ 29 and pO₂ 76; pCO₂ 26 and pO₂ 78). (DX 67). The September 25, 1996 study was non-qualifying at rest (pCO₂ 35.0 and pO₂ 88). (CX 2). The August 27, 1997 test was non-qualifying at rest (pCO₂ 35 and pO₂ 88), but became qualifying with exercise with a drop in the pO₂ (pCO₂ 30.3 and pO₂ 69). (DX 92). The February 17, 2000 study was qualifying at rest (pCO₂ 30.0 and pO₂ 70.0). (CX 1).

¹⁰ Claimant had undergone a stress test completed by Dr. Barghouthi which revealed that “the ejection fraction of the heart had dropped significantly to 33% and there was a moderate inferior wall perfusion defect compatible with ischemia. He judged the overall cardiac ejection fraction as 43%.” (Ranger EX 11).

Dr. Castle, Dr. Zaldivar, Dr. Fino and Dr. Jarboe also all positively linked the reduced diffusing capacity to Claimant's smoking history. Each doctor alludes to the fact that cigarette smoking commonly causes a reduction in the diffusing capacity, and that Claimant was actively smoking throughout his pulmonary function tests.

Additionally, Dr. Jarboe specifically opined that Claimant's mild to moderate reduction in diffusion is the result of pulmonary emphysema. While Dr. Jarboe acknowledged that coal dust inhalation does cause mild focal emphysema, Claimant had no evidence of significant dust retention of the chest radiograph, prompting Dr. Jarboe to positively conclude that Claimant's emphysema is ascribed to smoking. (DX 111). Dr. Castle positively linked Claimant's chronic bronchitis to his tobacco use. (DX 92). Because the medical opinions of Drs. Fino, Jarboe, Castle and Zalidvar linking Claimant's respiratory ailments to his smoking alone are thoroughly explained and well-documented, I find that they are entitled to more weight.

Finally, the qualifications of the physicians are relevant in assessing the respective probative values to which their opinions are entitled. *Burns v Director, OWCP*, 7 B.L.R. 1-597 (1984). I place greater weight on the opinions of Drs. Jarboe, Fino, Zalidvar, and Castle because of their superior qualifications: they are all board certified in internal medicine and pulmonary medicine. *Scott v. Mason Coal Co.*, 14 BLR 1-38 (1990). On the other hand, Dr. Rasmussen is a board certified internist.

As I have now accorded more weight to the opinions of physicians who positively linked Claimant's COPD to his tobacco use, and I have found no credible diagnosis of clinical or legal pneumoconiosis, I find that Claimant has failed to establish, by a preponderance of the evidence, the existence of pneumoconiosis under §718.202(a)(4).

Because the Claimant has failed to establish the existence of pneumoconiosis under any of the provisions of §718.202(a), he cannot be found to be entitled to federal black lung benefits. As such, it is unnecessary to discuss the additional issue on remand of whether the Claimant has established that he is totally disabled due to pneumoconiosis.

ORDER

Based upon the foregoing, the claim of Marvin Dan Kingery for benefits under the Black Lung Benefits Act is Denied.

A

RICHARD E. HUDDLESTON
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this decision and order may appeal it to the Benefits Review Board within 30 days from the date of this decision and order, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances